

342 and 403 respectively were not kept, 23.1% v. 27%,  $p < 0.05$ . Four hundred and seventy patients (217 males and 253 females) were responsible for these failed appointments. However, 243 (104 males and 139 females) returned to the clinic and were interviewed.

The diagnosis among the 470 defaulters were genital warts 132 (28.1%); *Chlamydia trachomatis* infection 35 (7.4%); Non-specific urethritis 29 (6.2%); genital herpes 10 (2.1%); gonorrhoea 9 (1.9%); *Trichomonas vaginalis* infection 6 (1.3%); positive syphilis serology 2 (0.4%). A large group of "Others" included patients with candidiasis, balanitis, scabies etc. When the incidence of different sexually transmitted diseases among defaulters was compared with that of non-defaulters, the only significant difference was that genital warts were commoner among defaulters 28.1% v. 11.8%,  $p < 0.001$ . Among those interviewed the reasons for defaulting are shown in the table. There were significant differences between the men and the women. Four women were "afraid" and therefore defaulted. Two of them were pregnant and they thought that further examination would harm their pregnancies. The other two were sick after taking metronidazole tablets and were hence discouraged from making further visits. Out of the 470 defaulters, only two women who had chlamydia cervicitis did not receive appropriate treatment. All efforts by the health advisers to get them back to the clinic were unsuccessful.

The frequency of failed appointments varies between clinical departments; 4.8% reported from a diabetic clinic<sup>1</sup> and 35% in a paediatric clinic.<sup>4</sup> In our study, since only half of the defaulters came back, their reasons for defaulting cannot be generalised to all defaulters. Nevertheless, some lessons can be learnt from this exercise. The age distribution of defaulters was similar to that of our clinic attenders. Likewise, apart from genital warts, the incidence of many common sexually transmitted diseases was similar among defaulters and non-defaulters. An interpretation of this is that the problem is not peculiar to any age or diagnosis group. There were more defaulters from the morning sessions. This may relate to some of the reasons—work and domestic problems given by the patients. Alteration of clinic times may improve matters.

The importance and the need for a subsequent visit should be stressed by the clinic

staff to the patients. When subsequent appointments are being made, patients should be asked if the date and session are convenient. Secondly, if the patient later on finds out that the appointed time is inconvenient, he/she should be aware that a telephone call to arrange another date is a better alternative to a failed appointment. Identification of remediable causes of failed appointments from indepth analysis and then making appropriate adjustments will lead to efficient use of resources.

A A OPANEYE

Department of Genitourinary Medicine,  
Sunderland District General Hospital,  
Kayall Road, Sunderland SR4 7TP, UK

E PARKER

J BAILEY

M WALZMAN

A A WADE

Department of Genitourinary Medicine,  
Coventry & Warwickshire Hospital,  
Stoney Stanton Road,  
Coventry CV1 4FH, UK

Address correspondence to Dr AA Opaneye.

- 1 Scobie IN, Rafferty AB, Franks PC, Sonksen PH. Why patients were lost from follow-up at an urban diabetic clinic. *BMJ* 1983;286:189-90.
- 2 Dickey W, Morrow JL. Can out-patient non-attendance be predicted from the referral letter? An audit of default at neurology clinics. *J R Soc Med* 1991;84:662-3.
- 3 McGlade KJ, Bradley T, Murphy GJ, Lundy GP. Referrals to hospital by general practitioners: a study of compliance and communication. *BMJ* 1988;297:1246-8.
- 4 Andrews R, McNeish AS, Morgan JD, Addy DP. Understanding non-attendance in out-patient paediatric clinics. *Arch Dis Child* 1990;65:192-5.

Accepted for publication 7 May 1993.

## Carcinoma of the vulva and asymptomatic lichen sclerosus

Genitourinary medicine departments are seeing an increasing range of non-infectious problems. We report a patient with asymptomatic lichen sclerosus (LS) who presented with squamous cell carcinoma of the vulva.

A 39 year old female was referred by her general practitioner (GP) with a solitary perineal swelling of one month's duration which had developed three months after a casual sexual contact. Her GP made an initial diagnosis of an infected sebaceous cyst and prescribed antibiotics. She gave no history of sexually transmitted disease (STD), including genital warts and was otherwise asymptomatic.

On examination she was found to have LS of the anterior vulva (confirmed on punch biopsy). She was also noted to have a firm, discreet 1.5 × 1.0 cm raised nodule with a central warty area, lateral and anterior to the anal margin. There was no evidence of condylomata acuminata and no inguinal lymphadenopathy. Biopsy revealed a squamous cell carcinoma. She was referred to the gynaecology department for a wide local excision with block dissection of the inguinal lymph nodes. Histology confirmed complete excision of the tumour, with LS in the surrounding skin, and no evidence of node involvement.

The incidence of vulval carcinoma appears

Table Reasons why patients did not attend

	All patients 243 (%)	Men 104 (%)	Women 139 (%)	p value
Work	48 (19.8)	31 (29.80)	17 (12.20)	0.001
Forgot	56 (23)	26 (25.00)	30 (21.58)	NS
Holiday/travel	22 (9.1)	11 (10.58)	11 (7.91)	NS
Clerical error	24 (9.9)	10 (9.62)	14 (10.10)	NS
Felt well	17 (6.7)	9 (8.65)	8 (5.76)	NS
Domestic problems	29 (11.9)	7 (6.73)	22 (15.83)	0.025
Illness	27 (11.1)	6 (5.77)	21 (15.10)	0.025
Not asked	6 (2.5)	3 (2.88)	3 (2.16)	NS
School	10 (4.1)	1 (0.96)	9 (6.47)	0.025
Afraid	4 (1.7)	0	4 (2.88)	

to be increasing in young women and this change has been linked to the increasing prevalence of human papilloma virus infection and vulval intra-epithelial neoplasia (VIN).<sup>1</sup> A causal link is unproven but both LS and VIN may be associated with the development of carcinoma.<sup>2-4</sup> Published data have either assessed the skin changes in patients with a primary diagnosis of carcinoma of the vulva<sup>2-4</sup> or have observed cohorts of patients with LS for the development of carcinoma of the vulva.<sup>5,6</sup> Buscema *et al* in a series of 98 patients with vulval carcinoma identified four patients in whom LS was found adjacent to the carcinoma.<sup>3</sup> Zaino *et al* noted the presence of adjacent LS in 15 of 60 patients with vulval carcinoma.<sup>4</sup> Leibowitch *et al* described a high proportion of patients with vulval carcinoma associated with LS. In their series of 78 patients with vulval carcinoma 61% had LS identified histologically adjacent to the tumour. Many of these patients also had squamous hyperplasia or VIN III.<sup>2</sup>

The observation that LS may be present in association with a vulval carcinoma does not establish a cause and effect relationship. Studies in the 1950s and 1960s suggested that carcinoma of the vulva occurred in up to 10% of patients with LS. Of 465 patients with LS in published series 16 had coexistent carcinoma of the vulva.<sup>5</sup> The precise relationship between symptomatic and asymptomatic LS and the development of vulval carcinoma remains unclear. In Leibowitch's series of 78 patients with carcinoma of the vulva 43 patients had undiagnosed LS.<sup>2</sup>

This case is a reminder that squamous cell carcinoma should be remembered in the differential diagnosis of a solitary vulval swelling, particularly if there is associated LS. Aggressive treatment of LS with topical steroids and careful long-term follow-up should be instituted,<sup>7</sup> but whether this will reduce the incidence of vulval carcinoma remains an untested hypothesis.

I BYREN  
V VENNING  
A EDWARDS

Department of Genitourinary Medicine,  
Radcliffe Infirmary, Oxford OX2 6HE, UK

- 1 Andersen WA, Franquemont DW, Williams J, Taylor PT, Crum CP. Vulvar squamous cell carcinoma and papilloma viruses: two separate entities? *Am J Obstet Gynaecol* 1991;165:329-36.
- 2 Leibowitch M, Neill S, Pelisse M, Moyal-Baracco M. The epithelial changes associated with squamous cell carcinoma of the vulva: a review of the clinical, histological and viral findings in 78 women. *Br J Obstet Gynaecol* 1990;97:1135-9.
- 3 Buscema J, Stern J, Woodruff JD. The significance of the histologic alterations adjacent to invasive vulvar carcinoma. *Am J Obstet Gynaecol* 1980;137:902-9.
- 4 Zaino RJ, Husseinazadeh N, Nahhas W, Mortel R. Epithelial alterations in proximity to invasive squamous carcinoma of the vulva. *Int J Gynaecol Pathol* 1982;1:173-84.
- 5 Hart WR, Norns HJ, Helwig EB. Relation of lichen sclerosis et atrophicus of the vulva to development of carcinoma. *Obstet Gynaecol* 1975;45:369-77.
- 6 Meyrick Thomas RH, Ridley CM, McGibbon DH, Black MM. Lichen sclerosis et atrophicus and auto immunity—a study of 350 women. *Br J Dermatol* 1988;118:41-6.
- 7 Wojnarowska F, Dalziel KL, Millard P. The treatment of vulval lichen sclerosis with a very potent topical steroid (clobetasol propionate 0.05%). *Br J Dermatol* 1991;124:461-4.

Accepted for publication 11 May 1993.

## ***Staphylococcus aureus* pericarditis in a patient with AIDS**

Bacterial pericarditis is rare in patients with HIV infection. Recently we have seen a 36 year old homosexual man who had pericarditis due to *Staphylococcus aureus*.

The patient, who had never injected drugs, presented with a 24 hour history typical of acute pericarditis. On examination he was pyrexial with a temperature of 38.7°C and had a pericardial friction rub. A chest radiograph showed a normal sized heart and an ECG showed widespread concave ST segment elevation. An echocardiogram showed a moderate pericardial effusion and an indium-111 labelled human polyclonal immunoglobulin (<sup>111</sup>In-HIG) scan<sup>1</sup> showed pericardial accumulation, suggesting an infective aetiology.

He had been HIV-1 seropositive for three years and *Pneumocystis carinii* pneumonia and cutaneous Kaposi's sarcoma were diagnosed 15 months before this admission. Eight weeks before presenting with pericarditis the patient had bronchitis due to *Haemophilus influenzae* and *Streptococcus pneumoniae*; at this time fiberoptic bronchoscopy had confirmed endobronchial Kaposi's sarcoma. His CD<sub>4</sub> count was 0.04 (normal range 0.35–2.20) × 10<sup>9</sup>/l. Chemotherapy with vincristine 2mg and bleomycin 30 mg once every three weeks was commenced.

At the time of his admission with pericarditis, further results showed a white blood cell count of 2.8 × 10<sup>9</sup>/l (69% granulocytes), no elevation of cardiac enzymes and no rise in viral titres in paired sera; Coxsackie B IgM was negative. Sputum samples grew *H. influenzae* and *Str. pneumoniae*, which were treated with tetracycline in conventional doses; blood cultures were negative. A chest radiograph taken four days after admission showed an increase in the cardiac diameter and a second echocardiogram showed a pericardial effusion with pericardial thickening and diastolic collapse of the right atrium, but not of the right ventricle. The diagnosis was thought to be of chronic pericardial effusion due to *H. influenzae* and further intervention was not thought to be required.

Sixteen days after admission signs of impaired right ventricular filling developed with pulsus paradoxus of 15 mm Hg. Repeat echocardiography showed no right ventricular diastolic collapse but there was evidence of restrictive cardiomyopathy, suggesting pericardial infiltration. Neither computerised tomography nor gated magnetic resonance imaging (MRI) showed evidence of pericardial Kaposi's sarcoma. On the 22nd day after admission pericardiocentesis was performed; Gram-stain of the pericardial aspirate demonstrated numerous pus cells, and culture of the fluid yielded a heavy pure growth of *Staphylococcus aureus*, sensitive to penicillin. Prolonged culture for mycobacteria was negative and cytology revealed no malignant cells. Despite IV teicoplanin and oral rifampicin he developed increasing peripheral oedema and